MAN AT HIGH SUSTAINED +Gz ACCELERATION

R. R. Burton, et al

Advisory Group for Aerospace Research and Development
Paris, France

March 1974

AGARD-AG-190

AGARD-AG-190

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on

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and E.D.Michaelson

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AGARDograph No.190

MAN AT HIGH SUSTAINED +Gz ACCELERATION

by

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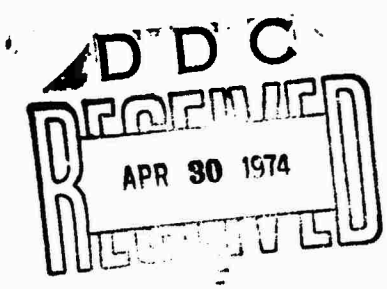

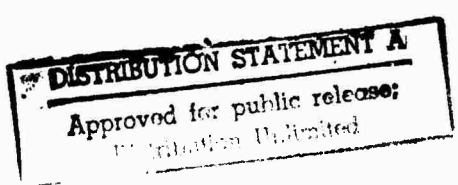

This AGARDograph was sponsored by the Aerospace Medical Panel of AGARD.

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Published March 1974

612.014.4 : 531.113 : 629.7.076.2

Printed by Technical Editing and Reproduction Ltd
Harford House, 7–9 Charlotte St, London. W1P 1HD

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ACKNOWLEDGEMENTS

The authors acknowledge the technical help of those persons listed below who were responsible for the operation of the centrifuge, instrumentation of volunteers, and collection of data. The human volunteers who served as experimental subjects in these studies were dedicated persons who patiently tolerated many uncomfortable situations. In addition, we thank Mrs. Joanne Rigdon for typing this manuscript (both rough draft and final copy)--much of which was performed during the 1973 Christmas holiday.

Finally, we would like to express our appreciation for the critical evaluation of the manuscript by Drs. F. W. Baumgardner, USAF School of Aerospace Medicine, and E. H. Wood, the Mayo Clinic. Dr. Wood served as the official technical reader of the manuscript and both he and Dr. Baumgardner offered many excellent suggestions for improving the text of the manuscript.

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MAN AT HIGH SUSTAINED $+G_z$ ACCELERATION

R. R. Burton, S. D. Leverett, Jr., and E. D. Michaelson

High Sustained $+G_z$ Defined

High sustained $+G_z$ acceleration (HSG), as used in this text, will include positive acceleration exposures of 6 G and greater for periods longer than 15 sec. These levels and durations of $+G_z$, generally speaking, require that an air crew member perform repeated straining maneuvers in combination with an anti-G suit in order to maintain vision. Many individuals in a relaxed state can tolerate acceleration levels less than 6 G with the aid of an anti-G suit. High G levels (+6 to 10 G_z) of short duration may be tolerated by the use of almost any type of straining effort requiring only minimal coordinated skeletal muscular tensing.

At present, there is much interest in HSG; for the low wing loading and high thrust to weight ratios of recently developed aircraft suggest that they will have HSG capabilities which approach or may even exceed man's "tolerance" to $+G_z$. It is important, therefore, to define the limits of man to HSG and to examine his physiology during HSG exposure. This research will not only indicate the problems which will surely exist during exposure to accelerative levels of HSG magnitudes and durations, but should suggest methods to alleviate or at least modify difficulties thus encountered by man.

This information on HSG, however, appears to be more than an extension of the present knowledge of man at acceleration levels (less than 6 G for short durations) because in many ways HSG is a different environment with respect to modifying the physiology of man and the utilization of anti-G techniques. Consequently, much of the information available regarding human exposure to accelerations is not applicable to HSG. This is unfortunate because much is known concerning man at lower G levels and several excellent reviews are available (1, 2, 3, 4). However, because similar nomenclature is used for all acceleration studies, it is assumed that the reader has been exposed to the information contained in these review articles.

Although HSG research has just begun, the authors decided that an early review would serve several purposes: (a) to alert the scientific community that a new environment "tolerable" for man exists; (b) to collate the beginning scientific efforts, in this field, which may suggest the proper direction of continuing HSG investigations; and (c) to use this initial information as a basis for "human factors" in aircraft design, and as a training program for personnel using high performance aircraft.

Establishment of Human Limits to HSG

The word "tolerance" is used in acceleration research to identify the G level at which specific physiologic systems fail. Criteria used in quantifying acceleration tolerance commonly are of visual origin--various stages of light loss: peripheral light loss (PLL), central light dim (CLD), and central light loss (CLL). However, other criteria (such as auditory phenomena, cardiorespiratory responses, and consciousness) also have been used in acceleration studies (5).

Using visual criteria (PLL/CLD), Parkhurst et al. (6) designed an experiment to determine human "tolerance" limits to HSG. They wisely set a G level/duration exposure maximum limit--3 G for 45 sec--and rather surprisingly, the majority of subjects were able to tolerate this without reaching the visual criteria of PLL/CLD. One subject of the Parkhurst study told an investigator in our HSG research group that he could probably "tolerate without loss of vision" a maximum of +11 G_z for at least 1 min. In the same study, Shubrooks (7) examined the electrocardiograms of these subjects and reported several types of cardiac arrhythmias which suggested moderate heart stress during HSG. Because maximum G limits for man cannot be established, "tolerance" to HSG is currently measured as "enduring" levels and durations of G exposure: (a) with a maximum of 100% peripheral light loss (PLL) and 50% central light loss--central light dim (CLD); (b) without the occurrence of "serious" cardiac rhythm disturbance; (c) and without complete subject fatigue.

However, these criteria are quite inadequate in considering the possible hazards to the subject (whether or not G protection is provided) during exposure to HSG where man's tolerance may surpass his ability to defend himself adequately against environmental conditions that could eventually lead to a pathologic situation. Also, criteria are needed which measure human performance at HSG, since survival only is not enough; man will also have to perform during HSG. Consequently, ideal criteria of human tolerance to G should measure performance as well as the physiological protection afforded the subject by the various anti-G methods used to attain HSG. Unfortunately, adequate criteria of HSG tolerance are not presently available.

Summary of Research Studies at HSG

Since the time of the tolerance study of Parkhurst et al. (6), several investigations have been conducted at the USAF School of Aerospace Medicine (SAM) to examine the physiology and potential pathology of man at HSG, and the utility of various methods available to increase human tolerance to G.

Shubrooks and Leverett (8) studies G-suited subjects with both 15- and 45-sec rapid onset rate (ROR; 1 G/sec) G profiles and compared the efficacy of the M-1 and Valsalva as anti-G maneuvers. In another study of anti-G methods, Shubrooks (9) compared cardiovascular responses of the M-1 maneuver with

continuous positive pressure breathing (PPB) and demonstrated that PPB provided an increase in G tolerance equivalent to that obtained with the M-1 maneuver and with less subject fatigue.

The information obtained in the foregoing studies were extended with more detailed physiologic measurements obtained during an investigation shared by SAM, at Brooks Air Force Base, Texas, and the Royal Air Force (RAF) Institute of Aviation Medicine (IAM) at Farnborough, Hants, England. In this SAM/IAM investigation, volunteer subjects were exposed to accelerations up to +8 G_z for 60 sec. Two separate studies were involved: (a) The USAF-CSU-12/P anti-G garment was compared with the RAF "mini" anti-G suit, and (b) the M-1 maneuver was evaluated and compared with PPB at the same G levels. The numerous physiologic measurements included: direct eye-level arterial pressure; central venous pressure; esophageal and gastric pressures; suit and mask pressures; and ankle venous pressure. Arterial blood gas and pH measurements, A-P chest X-rays, and electrocardiographic data were also obtained. The results of this investigation and their interpretation have been published as USAFSAM-TR-73-21 (Leverett et al., 10).

Michaelson (11) who studied nine subjects at levels up to +7 G_z for 45 sec obtained measurements of arterial blood, expired gases, and ventilation. These data, along with calculated indices of pulmonary gas exchange, are discussed and compared here with the measurements obtained in the SAM/IAM study.

A unique electroencephalography study, during 45 sec exposures of 4.5 to 7 G, was reported by Berkhout et al. (12). Using the technique of spectral analysis, they identified EEG changes associated with HSG.

The adverse effects of the combined stress of alcohol and acceleration (13) and cigarette smoking and acceleration (14) have been measured using, respectively, performance and spirometry.

The tilt-back seat as a mechanism of increasing G-tolerance has been examined by Burns (15) and Rogers et al. (16) who demonstrated that up to 10 G, acceleration tolerance was significantly improved (compared to control levels) at seat back-angles greater than 30 degrees.

Because of severe physiological stresses imposed by HSG and because of requirements for complex invasive techniques for measuring physiologic changes, certain studies cannot be safely performed using human subjects. Consequently, animal studies are important in examining pathology and physiology at HSG. In this regard, Burton (17) reported that the miniature swine served as an adequate animal model for pathophysiology studies at HSG.

The studies just cited indicate that the physiologic effects of G and resulting performance decrements are profound and that many problems exist. Presently, more information is needed on the pathophysiological effects of repetitive G exposures, and of G profiles that more closely simulate an air-combat maneuvering environment. Also, additional studies are planned to define the safe tolerance of man to HSG.

Exposing Man to HSG

Human tolerance to HSG is attained by combining various anti-G methods each of which (with its specific effects) increases acceleration tolerance; i.e., the combination of anti-G benefits results in the ability of man to tolerate HSG. The basis of HSG tolerance is man's inherent ability to resist, while relaxed, the changes in his physiology resulting from G exposure--basically a manifestation of several integrated cardiovascular responses. This relaxed tolerance is usually measured as the G-level at which loss of vision occurs. This innate resistance to G--which is an individual characteristic (18)--may be supplemented by using various (a) mechanical aids and/or (b) conscious physiologic countermeasures which increase G tolerance. The former would characterize anti-G suits and the latter would be voluntary physical efforts (straining maneuvers).

Anti-G Suits

Two types of anti-G suits have been used in HSG research--the 5-bladder interconnected inflatable anti-G suit (USAF CSU-12/P), and the 3-bladder interconnected inflatable mini anti-G suit (RAF prototype; Fig. 1). The mini-suit, basically, is the standard 5-bladder suit modified by removing the two calf bladders. The British were particularly interested in this suit for its greater comfort in the hotter tropical climates and for its ease in donning. Relaxed G tolerances are similar for both suits (19).

Both suits are similarly inflatable, and use anti-G valves which begin suit inflation at approximately 2 G and continue the inflation as a rectilinear function of G at a rate of 1.5 psi/G, with a maximum suit pressure of 10.5 psi.

Voluntary Straining Maneuvers

In order to maintain normal vision at higher G levels, an individual wearing an inflatable anti-G suit must perform some voluntary straining (forced pulmonary exhalation--muscular tensing) type of maneuver. This straining effort is usually required at +6 G_z and above. In the 1940's, Wood and his co-workers discovered that voluntary straining maneuvers--which they called the M-1 and M-2--when begun just prior to and continued during acceleration exposure, would increase the G level at which visual dimming began (20,

21, 22, 23). Since that time, several reports have appeared in the literature describing modifications of the original M-1 and M-2 maneuvers and the anti-G benefits arising therefrom (Table I)

Photo A

Photo B

Fig. 1: The RAF prototype "mini-suit" (photo A)--compared with a standard USAF multibladder G-suit, CSU-12/P (photo B). (The major difference is the absence of calf bladders in the mini-G-suit.) The subject also was fitted with the RAF soft helmet and P/Q oxygen mask (ref. 10).

Table I. Several types of voluntary straining maneuvers are compared regarding methods of performance and increased G provided.* (Note: References are in parentheses.)

			Tensing of:				
Name of Anti-G Effort	Forced Exhalation	Closed Glottis	Abdominal Muscles	Skeletal Muscles	Repeated During G	Increased G* w/o G-suit	Increased G* with G-suit
M-1[1]	Yes	Partially	Yes	Yes	Yes	2.4(21);2.5(22); 1.9(17)	3.4+(6); 3.0+(8)
M-2[2]	Yes	Yes	Probably[3]	No	No	1.3(21);None(22)	---
L-1[4]	Yes	Yes	Yes	Yes	Yes	0.9+(8)	1.5+(8); 3.0+(8)
Straining[5]	Probably	Probably	Yes	Yes	Probably	1.1(24)	1.0(24)
Arm Muscle Contraction	No	NA	No	Yes[6]	NA	0.9(25)	0.6(25); 0.6(22)
Leg Muscle Contraction	No	NA	No	Yes	NA	0.2(26)	---

* Increased G is considered as the increase in G tolerance over relaxed G tolerance.
[1] M-1 apparently indicates straining maneuver number 1; [2] M-2 is frequently called the Valsalva experiment or Valsalva maneuver--originally the M-2 referred to "blowing against a closed manometer system so as to maintain intrapulmonary pressures of 40 to 60 mm Hg for 10 sec immediately before g exposure" (ref 21); [3] It is difficult to force exhale without tensing the abdominal muscles; [4] Also called a modified Valsalva maneuver; [5] The word straining appears in the literature without reference to the type of respiratory effort used by the subject; [6] Right arm only; NA = not appropriate.

Practically speaking, however, the most effective voluntary anti-G maneuver for HSG is a combination of three specific efforts: (a) pulling the head down between the shoulders to shorten the eye-heart distance (crouch position); (b) tensing peripheral skeletal and abdominal musculature as much as possible to support the diaphragm and heart, reduce venous space and increase vascular resistance thereby lessening blood pooling; and (c) increase intrathoracic pressure by a method of forced exhalation which raises arterial blood pressure at eye-level. The subject has a choice of two methods: (a) M-1 or (b) L-1. If high G is to continue for longer than a few seconds (sustained period of time), then the respiratory phase of the maneuver will have to be repeated at 3 to 5 sec intervals using rapid "inhalations," viz, approximately 1-sec "gasps." It is

important that this gasp period be performed as quickly as possible because during the gasp, eye-level arterial blood pressure is very low and if prolonged, CLL or unconsciousness will intervene.

In performing the M-1 maneuver, the gasp is comprised only of the inhalation phase. Exhalation is accomplished by slowly reducing lung volume with forced exhalation against a partially closed glottis, thus maintaining increased intrathoracic pressure. Some persons, however, find this effort irritates their throat so they force "exhale" against a completely closed glottis. This maneuver, called the L-1, requires slightly longer gasp periods because lung volume is not reduced during the so-called "exhalation" phase since the person's glottis is completely closed; i.e., the gasp period in the L-1 must include exhalation as well as inhalation. However, this gasp period can be accomplished rapidly (1 sec or less), therefore the L-1 is as effective in increasing acceleration tolerance at HSG as the M-1 (18). It is emphasized here that both the M-1 and L-1 require the crouch position and muscular tensing, if either is to significantly increase G tolerance.

Selection and Training of Subjects

At SAM, subjects who frequently participate in acceleration studies are grouped as high- or low-G riders according to their ability to tolerate more than 7 G for 1 min while wearing an anti-G suit and performing either the M-1 or the L-1.

All centrifuge subjects are healthy (must pass a USAF Class II flying physical examination), 18 to 40 years of age, and usually males. As they accommodate to the centrifuge and as their relaxed tolerances to acceleration are determined, it becomes readily apparent that some of these subjects will be capable of tolerating HSG. Selection criteria are difficult to determine, however, two factors--natural G tolerance and motivation--appear to predominate. Motivation includes more than desire. The subject must not only want to do it, he knows he will do it! With such an individual, proper training, and experience, a high-G rider will usually result. A natural high-G relaxed tolerance is an asset at HSG, although not a requirement. Because such a person does not require a maximum M-1 effort to maintain vision, he is less likely to become exhausted (since all voluntary straining maneuvers are extremely tiring). However, as shown in Figure 2, men with low relaxed G tolerances also can tolerate HSG.

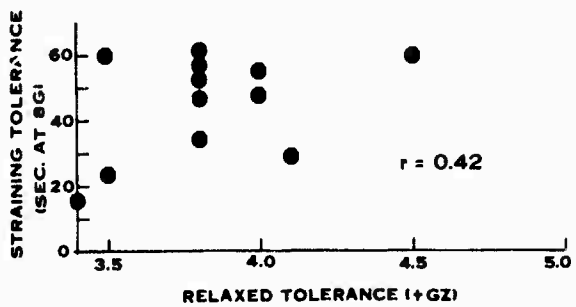


Fig. 2: Statistically insignificant correlation (correlation coefficient; r = 0.42) between relaxed G tolerances and straining tolerances (sec) at 8 G in the same subjects (ref 45).

Although individuals have become high-G riders, it is felt necessary to subject them, at least at biweekly sessions, to HSG (viz, 7 G for 30 sec). Experienced high-G personnel who are highly motivated can rapidly learn how to increase their duration of HSG tolerance; i.e., approximately 100 Tactical Air Command (TAC) fighter pilots using their anti-G suits have readily attained 8 to 8.5 G for 30 sec after a brief oral and visual training session on the technique of straining maneuvers.

Human Tolerance to HSG

To 14 selected subjects wearing anti-G suits, Parkhurst et al. (6) gave weekly HSG (45 sec) centrifuge exposures--beginning at 6.5 G and increasing by 0.5 G per week until 9.0 G was finally attempted by nine of the subjects. Of the five subjects (of the original 14) who did not attain 9 G, three reached 8.5 G, one sustained 7.5 G for 45 sec, and one was eliminated from the study because of the occurrence of transient ventricular tachycardia. (See cardiovascular section entitled "electrocardiogram.") These subjects did not attempt 9 G because they decided that they could not endure an additional 0.5 G. Since 9 G for 45 sec was the arbitrary maximum acceleration exposure allowed and because nine of the 14 subjects attained the maximum, we must conclude that man can tolerate greater than 9 G or 9 G for longer than 45 sec. The fact that all 14 subjects were able to achieve 7.5 G/45 sec is interesting as a significant increase in time and intensity at G compared with previous reports.

High sustained G tolerances of subjects using the mini-suit were compared with those of subjects wearing the CSU-12/P anti-G suit (10). HSG tolerance, as usual, was measured as the duration of time a person could maintain vision (at least PLL/CLD) at 3, 6, and 8 G with a maximum exposure of 1 min at each acceleration level. Each man tested both anti-G suits. The results of these runs are shown in Table II. All subjects tolerated 6 G for 60 sec; however, at 8 G, several persons failed to complete the entire 1 min run. Reasons for terminating the 8 G runs were easily characterized and are grouped as (a) subject fatigue or (b) loss of vision. Subject fatigue occurred at a mean time of 44 sec at 8 G whereas PLL/CLD was found much

earlier in the run at 28 sec. The G tolerances were not significantly different (according to paired t-tests) between anti-G suits.

Table II: Time (sec) spent (mean ± std. dev.) at 6 and 8 G by each subject wearing either the RAF mini-suit or the USAF CSU-12/P anti-G suit. All subjects had previously tolerated 3 G for 60 sec. One minute at each level was the maximum exposure time allowed. Statistical analyses (paired t-testing) showed no significant difference in tolerances between the suits (ref 10).

Subjects	6 G		8 G	
	Mini	CSU-12/P	Mini	CSU-12/P
RD	60	60	60	60
GD	60	60	48	60
JB	60	60	60	60
WM	60	60	30	18
SS	60	60	60	8
LP	60	60	60	46
Mean	60	60	54.5	42.0
Std. dev.	--	--	8.98	23.3

Several physiologic parameters were examined during these acceleration exposures, including: (a) heart rate; (b) electrocardiogram (ECG); (c) eye-level arterial blood pressure; (d) esophageal pressures; (e) central venous pressures; (f) lower body superficial venous pressure; and (g) heart mass size changes. The effects of high sustained G on these parameters are considered in detail later in this text; however, these parameters were not differentially affected by the type of anti-G suit worn.

The only major difference between these two suits was found at 6 G, at which level those men wearing the mini-suit complained of severe pain in the lower leg (calf) region. This pain was described by the subjects as characteristically "cramp-like." The calf pain usually occurred abruptly 5 - 10 sec into the run, and continued unabated until the acceleration intensity was reduced. Occasionally, however, the pain continued for several minutes after high G exposure, and some edema and tenderness in the calf region were apparent. Interestingly, and quite unexpectedly, leg pain in the same subjects wearing the mini-suit at 8 G was frequently absent or considerably reduced from that experienced at 6 G.

G-Tolerance Benefits of Positive Pressure Breathing (PPB)

PPB has been considered by several investigators as an aid for increasing G tolerance (4, 9, 22, 27, 28). Wood and Lambert (22) concluded that pressure breathing had "no appreciable effect" on tolerance to G, but the "protective value" of the G-suit was greater when the subject pressure breathed than when breathing normally. (They did not elaborate.)

Shubrooks (9) examined continuous PPB as a technique to increase tolerance during positive acceleration in three groups of subjects. He found that PPB was as effective as the M-1 in increasing either relaxed or straining G tolerances and was less fatiguing to the subjects. He concluded, however, that training, muscular tensing, and use of the anti-G suit were important in increasing the effectiveness of PPB against increased G.

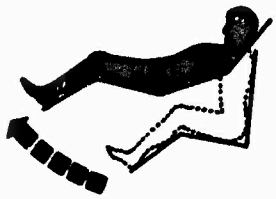
Recently, Leverett et al. (10) examined the value of continuous PPB at HSG by exposing six subjects (wearing a CSU-12/P anti-G suit) to a series of 1-min interrupted exposures of 3, 6, and 8 G. PPB was compared with the M-1, using the same subjects. The duration of each G exposure for each subject is shown in Table III. No significant differences (paired t-testing) regarding tolerable time at 6 or 8 G were found between the M-1 and PPB, thus identifying PPB as an effective method of increasing $+G_z$ tolerance. The majority of the subjects preferred PPB to the M-1 because of less fatigue with PPB, as earlier observed by Shubrooks (9).

Reclining Seat as an Anti-G Device

It is well known that a subject's G tolerance will be increased by changing his position with respect to the direction (vector) of the acceleration force to shorten his eye-aortic arch distance (15, 16, 29, 30). This effect occurs as the head is lowered (reclining seat), or as the pelvis and legs are elevated (Fig. 3). In either situation, the back angle of the subject increases relative to the $+G_z$ vector--going from the standard seated posture of 13° back angle to a 75° maximum.

The effect of this change in subject posture on relaxed G tolerance (visual criteria) of short duration (15 sec) has been determined by Burns (15) and is shown in Table IV. No change in G tolerance is found at 30°; but, at angles of 45° and greater, significant increases in G tolerance occur. It is of interest that flexion of the knees, elevation or depression of the legs above, at, or below heart level without elevation of the pelvis relative to heart and head location, does not alter G tolerance (15, 29).

TILT-BACK SEAT



PALE (PELVIS AND LEGS ELEVATING) SEAT

Fig 3: Tilt-back seat compared with the PALE seat (ref 46).

Table III: Duration in sec tolerated at 6 or 8 G by 6 subjects wearing the CSU-12/P anti-G suit and doing either the M-1 or PPB. All subjects had previously tolerated 3 G for 60 sec. One minute at each G level was the maximum exposure time allowed. Statistical analyses (paired t-testing) showed no significance difference in tolerance between the M-1 and PPB (ref 10).

Subjects	6 G		8 G	
	M-1	PPB	M-1	PPB
RS	33*	60	50	43
HL	60	60	38	56
JM	60	39	20	6
RL	60	60	14	32
TP	60	60	53	60
RZ	60	60	60	60
Mean	55.5	56.5	39.2	43.2
Std. dev.	11.0	8.57	18.7	20.5

* Inlet hose to mask occluded and prevented breathing.

Table IV: The influence of seat-back angle on relaxed G tolerance (ref 15).

Subjects	Relaxed G tolerance					
	13°	30°	45°	55°	65°	75°
1	4.0	4.0	---	5.3	6.2	8.0
2	4.4	4.0	4.8	5.0	7.4	8.5
3	3.5	3.7	4.3	5.5	6.3	7.0
4	3.5	3.5	4.2	4.9	5.9	7.3
5	3.5	3.5	4.3	4.9	7.0	10.0
6	4.9	4.5	4.5	5.0	7.5	8.0
7	4.0	4.3	4.8	5.2	6.1	7.0
Mean	3.97	3.93	4.48[a]	5.11[b]	6.48[c]	7.97[c]
±Std. error	0.20	0.15	0.11	0.09	0.24	0.40

Significantly different from values at 13°: a = P < .05; b = P < .005; c = P < .001.

Seat angles above 45° produce greater increases in G tolerance as the angle becomes greater; e.g., between 45° and 55°, a tolerance increase of 0.063 G/degree occurs whereas, between 65° and 75°, an increase of 0.149 G/degree occurs. Qualitatively, this type of response is characteristic of sinusoidal functions--an increasingly rapid reduction in eye-aortic arch distance as the angle of the subject's body, with respect to the G vector, becomes greater. Crossley and Glaister (31), in an earlier tilt-back seat study, also reported significant correlations between G tolerances (visual criteria) of relaxed subjects with eye-heart distance of 1/sine of the back angle. They also noted that a standard inflatable anti-G suit increased relaxed G tolerances of persons in the reclining seat even at tilt-back angles as great as 75°.

The back angle of 65° appears to offer the optimal seat modification for high acceleration aircraft--providing an increase in relaxed G tolerance of 65% and still permitting the pilot to have adequate vision in front of the aircraft.

The effect of reclining the seat, in relation to subject performance, has been examined by Rogers et al. (16). The seat angle was varied between 30° and 65°; and the subjects, during G exposures of approximately 1 min, responded to a tracking-target-task using a closed-loop subject-controlled centrifuge. The respective subject's performance was scored as percent target "hit" score at various G levels and seat angles. Significant improvements in task-score were reported for those subjects at seat angles of 45° and 65°, as compared with their achievements at 30° (Fig. 4).

Pathology Associated with HSG

Previous reports concerned with the occurrence of pathology in humans during $+G_z$ exposures have suggested a low incidence of minor adverse effects including: temporary vision loss, chest pain, dyspnea, motion sickness, non-pathologic changes in the electrical activity of the brain, various cardiac arrhythmias, generally considered to be non-serious, and unconsciousness (7, 12, 32, 33). These acceleration exposures were predominantly of short duration and at relatively low G (e.g., 6 G or less); although, Wood et al. (33) reported 2% to 3% of their total exposure time was at 6 G or above--apparently some exposures were longer than 15 sec--and Lambert and Wood (20) noted that in their studies "many persons" tolerated "sustained" exposures to +9 G_z (they did not specify the exact duration of exposure).

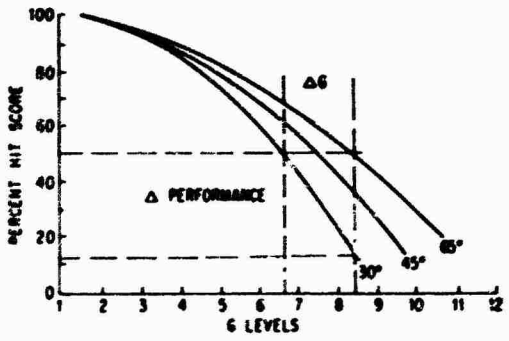


Fig 4: Acceleration tolerance (ΔG) and performance (Δ performance) at G are increased with greater seat back-angles (ref 16).

Apparently, the only serious pathology attributed to high G which has been reported in the literature is in an abstract in which a 9 G exposure, in an uncontrolled aircraft, is related to some form of temporary brain damage--symptoms of which disappeared within six months (34).

The occurrence of pathology in a "human analog" (an experimental animal with HSG responses similar to man) exposed to HSG tolerable to both man and the experimental animal model, has been investigated using adult miniature swine (17). Conscious pigs serve as adequate human acceleration models because they: (a) have the same vascular hydrostatic column length as man; (b) perform an M-1 like maneuver during G which results in arterial pressure responses at eye-level similar to those of man* and (c) have similar acceleration tolerance levels with and without anti-G suit inflations, as established for man (Table V).

Table V: Acceleration tolerances of man compared with those of adult miniature swine. Shown are mean values ± standard errors (range in parentheses) (ref 17).

	Without M-1		With M-1	
	No G suit	G suit*	No G suit	G suit*
Man				
PLL	3.7 ± 0.10†	5.4 ± 0.13	5.6 ± 0.17	8.8 ± 0.12
	(3.3 - 4.3)	(4.9 - 6.2)	(4.6 - 6.2)	(7.5 - 9.0)
CLL	4.7 ± 0.8	6.4	---	---
	(2.7 - 7.8)			
Swine				
PLL	4.1	5.7	5.1	7.6
CLL	4.9	6.6	6.0	8.6

* CSU-12/P anti-G suit; the anti-G suit used on the swine consists of the abdominal bladder (only) of the CSU-12/P anti-G suit; i.e., no suit counterpressure over the legs is available to the pig.

† Mean ± standard error.

Burton and MacKenzie (35) exposed six adult miniature swine, wearing inflated anti-G suits, to a series of +5, 7, and 9 G_z runs of 45 sec each. This series of acceleration exposures is similar to those of Parkhurst et al. (6) who exposed humans to HSG. Immediately after these G runs, the pigs were euthanized with an overdose of pentobarbital i.v., and were then autopsied. Grossly, the endocardium of the left ventricles showed evidence of recent hemorrhage of varying severity (Fig. 5). The degree and location of the endocardial hemorrhage was quantitated by grading the area of the left ventricle involved--1 (slight) to 3 (extensive); e.g., an example of 3 is visible in Figure 5. Of the 6 pigs autopsied, the mean score for papillary muscle hemorrhage was 2.3, and the extent of ventricular wall involvement was graded at 2.5. Histologically, heart hemorrhage was limited to the subendocardial area primarily involving the space between heart muscle and the endocardium. Similar heart lesions have been reported to occur in goats after exposures to negative acceleration (36).

These same pigs also exhibited skeletal muscle hemorrhage in the superficial portions of the rear legs, below the inflated anti-G suit where suit counterpressures were not available. (The G-suit worn by the pigs is described in Table V.) In humans, a similar, but less severe lesion--cutaneous petechiasis of the lower (unsupported) body--occasionally occurs during HSG.

These animal experiments are relevant to human exposure to HSG only to the extent that HSG has been demonstrated to produce pathology in animals which exhibit several human similarities. Since HSG can

*Consult Fig. 20 (presented subsequently) for additional information.

obviously produce pathologies, several methods of detecting their occurrence in humans are being investigated at SAM. These investigations include tissue serum enzyme tests, detailed electrocardiography pre- and post-HSG exposure, several cardiovascular measurements, and standard clinical pathology urine and blood tests (37).

Cardiovascular Responses of Man to HSG

Heart Rate

Heart rate responses to HSG have been considered by Parkhurst et al. (6), Shubrooks (7), and Leverett et al. (10). At low G levels, heart rate is known to be directly correlated with the level and duration of $+G_z$ exposure. At HSG, however, a G:heart-rate relationship is less discernible; for maximum heart rates appear to be obtained at relatively low $+G_z$ intensities; viz, 7 G (Fig. 6).

Fig. 5: The mediai endocardial wall of the left ventricle of an adult miniature swine shows recent hemorrhage (well-defined black areas) involving both wall and papillary muscle (ref 35).

Parkhurst et al. (6) noted that approximately 10-20 sec before the onset of a HSG exposure an increase in heart rate occurred. The pre-acceleration heart rate was directly correlated with the G-level that the subject anticipated. This response appears to be primarily psychological in origin, and contributes to approximately half of the increase in heart rate found in men exposed to HSG (Fig. 6). Although these data are from non-pilot subjects, Leverett et al. (38) have reported similar pre-G responses in USAF pilots about to experience HSG exposure.

As noted earlier, in order for a man to tolerate HSG he must tense his skeletal muscles and perform the M-1, L-1, or PPB. The respective influence of the anti-G maneuvers (M-1 or L-1) and PPB on heart rate was examined by Leverett et al. (10) at 1 G, without an increased G effect. Performing the M-1 at 1 G caused a statistically significant increase in heart rate above resting levels (Fig. 7). This increased heart rate at 1 G was similar to that found in subjects wearing anti-G suits and experiencing 6 G. However, these subjects were requested to perform a maximum M-1 effort at 1 G which usually exceeded the straining effort necessary to maintain vision at 6 G. On the other hand, an increase in heart rate was found in subjects at 3 G not performing the M-1--an acceleration effect only. PPB at HSG causes an increase in heart rate similar to the M-1--except at 1 G, both peak in heart rate and period of recovery are less with PPB (10). Heart rate change at HSG therefore appears to have three bases: (a) M-1 or PPB, (b) acceleration, and (c) psychological.

Occasionally, during exposure to HSG, a decrease in heart rate will occur. This decrease was first noted by Shubrooks (7) who reported "high G bradycardia," (as he termed it) in 4 of 9 subjects beginning at 16 to 38 sec into runs of 6.5 to 9 G (Fig. 8). During a 7.5 G run, one subject had his heart rate slow from 160/min at 18 sec of G to 80/min at 30 sec into the same run. This rapid reduction in heart rate was due to slowing of the sinus pacemaker which Shubrooks (7) attributed to a reflex baroreceptor response to the high levels of arterial pressure resulting from the M-1 maneuver However, he suggested that "increased vasovagal activity leading to syncope, predisposed to by the decrease in cardiac filling" also may have been a factor. A direct correlation between a vasovagal reaction and subendocardial hemorrhage has been demonstrated in cats by Öberg and Thorén (39).

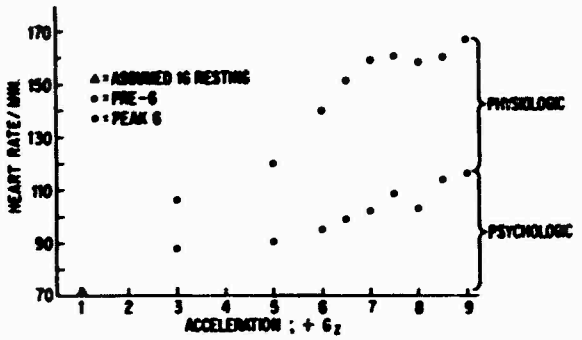


Fig. 6: The mean maximum heart rates from a group of 9 men are compared at various G levels of 45 sec duration, with their mean pre-G heart rates.

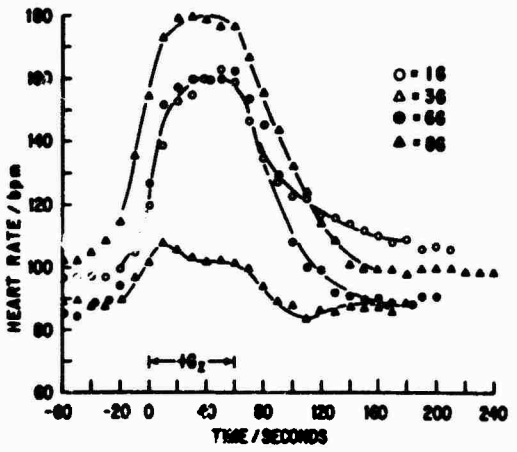


Fig. 7: Heart-rate responses (group means) to 60 sec of G with six subjects performing the M-1 at 1, 6, and 8 G. (The M-1 was not performed during the 3 G exposure.) From Leverett et al. (10).

Electrocardiogram

Cardiac arrhythmias frequently occur during and immediately after exposure to HSG; e.g., Shubrooks (7) found only 1 of 14 men exposed to HSG did not have any premature ventricular contractions (PVC). These premature beats tended to increase during the latter portion of the G exposure and appeared to be directly correlated with the inspiratory phase of the M-1. Also, it seems that PVC occur with increased frequency at the higher HSG levels--considering the occurrence of PVC in nine men who experienced exposures of 6.5 to 9 G in Shubrooks' study, their incidence rate (PVC/sec of exposure) was approximately twice as great at 9 G (0.22) than found at 7 G (0.10). Since heart rate is similar at 6.5 G and above, the incidence of PVC/HR is probably greater above 7 G--G levels where man has to perform a maximum M-1 to maintain vision. It was noted by Leverett et al. (10), however, that performing a maximum M-1 effort at 1 G (without the influence of HSG) does not produce cardiac arrhythmias.

All PVC found by Shubrooks showed abnormal QRS morphology, absence of P waves or evidence of retrograde ventriculoatrial conduction, and frequently a complete compensatory pause. Never did the PVC appear to have a parasystolic origin. Shubrooks also noted that the PVC were frequently multiform and occasionally occurred in runs of 4 to 7.

Leverett et al. (10) also reported the occurrence of similar cardiac arrhythmias in men during and after exposure to 8 G for 60 sec. Examples of these PVC are shown in Figure 9. Unlike Shubrooks (7), however, Leverett's group found no incidence of "long runs" (4 to 7) of PVC.

Possible causes of these abnormal beats, as discussed in detail by Shubrooks (7), are: high heart rate (possibly due to an excessive sympathetic activity); changes in heart position; and some degree of cardiac ischemia and/or possibly changes in cardiac filling and blood volumes. Owing to the possibility of myocardial ischemia or severely decreased heart volumes, it should be emphasized here that--although these cardiac rhythm changes are not "serious"--additional stresses could precipitate "a sustained serious rhythm disturbance (7)."

After exposure to HSG, a marked sinus arrhythmia occurs which appears to parallel a post-G arterial pressure peak phenomenon (10) (note this section, "Postacceleration Responses").

Because of ECG artifacts due to muscular tensing and electronic filtering, S-T segment and T-wave changes noted during HSG are difficult to evaluate. However, a decrease in T-wave amplitude and some S-T segment depression of the ischemic type have been observed during exposure to HSG. Shubrooks noted that subjects exhibiting the higher incidence of PVC also showed S-T segment depression. For more details regarding cardiac rhythm changes during HSG, consult Shubrooks (7).

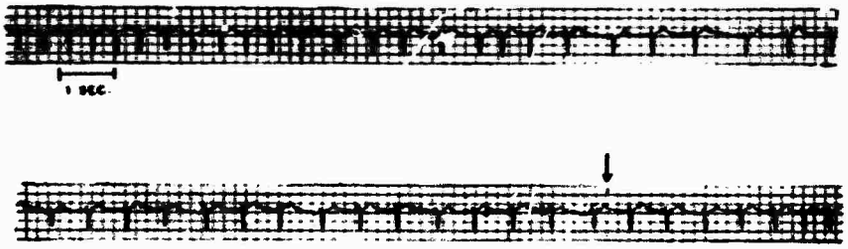


Fig. 8: ECG (sternal lead) during a 7.5 G run. Tracing is a continuous strip beginning 19 sec after 7.5 G was reached and continuing through deceleration; beginning of deceleration is indicated by arrow. This example of high G bradycardia is from Shubrooks (7).

Arterial Pressure

In relaxed subjects exposed to $+G_z$ levels where vision is lost, the arterial blood pressure (AP) at eye level can be rather accurately estimated--PLL occurs with a systolic AP of 50 mm Hg and CLL at 20 mm Hg (20). Such a cardiovascular response is typical of a simple hydrodynamic model such as that built and tested by Britton et al. (40). If the physical hydrostatic pressure influence could not be modified by various methods then man would lose vision and consciousness at +6 G_z (Fig. 10). However (as already noted in this text), man can tolerate without loss of vision more than +9 G_z for longer than 45 sec; i.e., systolic arterial blood pressure at eye level is probably greater than 50 mm Hg a considerable portion of the time at HSG.

Because various conscious efforts and anti-G techniques (M-1, L-1, PPB, etc.) and/or the anti-G suit are necessary to increase man's $+G_z$ tolerance to these high levels, their relationship to systolic arterial blood pressure at eye level is of great interest to acceleration physiologists and recently has been considered in three studies (8, 9, 10).

Anti-G Suit Effect

At HSG, the primary function of the anti-G suit is considered to be one of augmentation of leg muscle tensing and support of the diaphragm for performing the M-1 or Valsalva maneuver. However, it should not be forgotten that the anti-G suit affects the cardiovascular system of a relaxed person in a

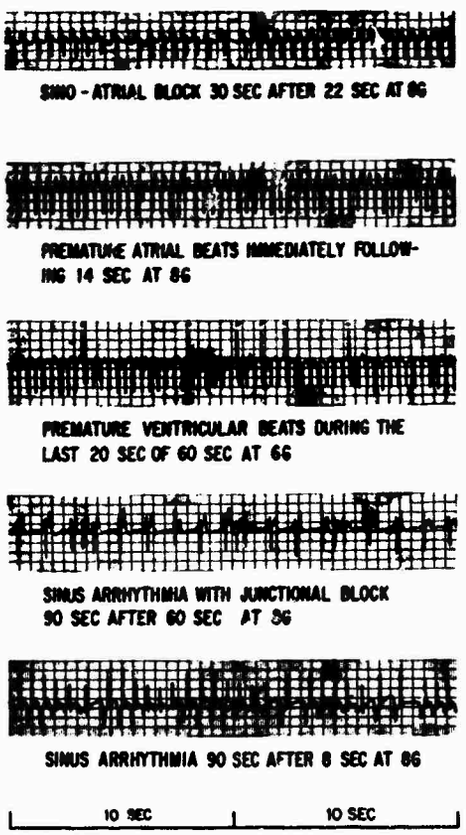


Fig. 9: Recording (sternal electrodes) of five types of cardiac arrhythmias observed during and after exposure to HSG; time-scale is shown at the bottom of the figure (ref 10).

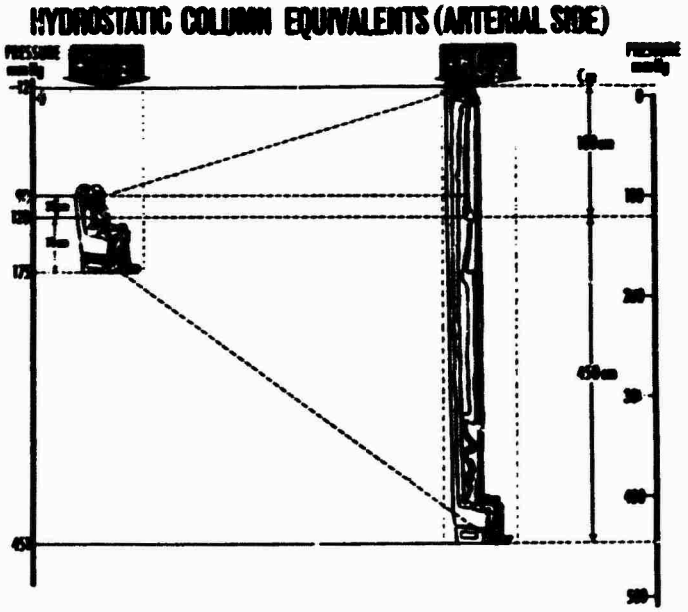


Fig. 10: Hydrostatic column effect in seated pilot at 1 G and at 6 G. Note the negative hydrostatic pressure developed at eye level if the calculated values (physical effect only) were used without considering physiological counter-maneuvers.

Fig. 11: Arterial eye-level pressure responses of a relaxed person with and without the anti-G suit (ref 8).

way that, by itself, increases G tolerance (visual criteria); viz, significantly increased eye-level arterial pressure (Fig. 11).

M-1 and L-1 Effects

Until recently, little was known regarding the cardiovascular response to either the M-1 or L-1 maneuver during HSG. Even at lower short-term G-levels physiologic effects of the L-1 maneuver were misunderstood. Since less noise and laryngeal irritation are associated with the L-1, many pilots appear to prefer it to the M-1; and therefore Shubrooks and Leverett (8) compared the effects of these two maneuvers upon specific cardiovascular parameters. Eye-level arterial pressure responses which fluctuated with breathing efforts were similar for both maneuvers at HSG, as shown in Figure 12. This common response included a gradual increase in end-diastolic pressure as the maneuvers were repeated during G exposure. They concluded that in conjunction with vigorous skeletal muscular tensing--with or without anti-G suit inflation-- the eye-level arterial responses and, therefore, increases in G tolerance for the M-1 or L-1 were equivalent.

The physiologic bases for these arterial pressure responses at HSG, regarding either maneuver, appeared to involve primarily a reduction in venous pooling from the lower extremities and an adequate venous return to the thorax because of the contraction of abdominal and peripheral skeletal musculature. This response occurs regardless of the fact that the intrathoracic pressure increases during either the M-1 or L-1 exhalation phases. These muscular contractions also effect a generalized sympathetic response via afferent neural stimuli (25). Other physiologic considerations regarding systemic arterial pressure at eye level would include: (a) decrease in the heart-to-head distance resulting from the ascent of the diaphragm during the exhalation phase of either maneuver; and (b) baroreceptor reflex responses. The latter consideration, although of primary importance in relaxed G tolerances, appears to be much less important at HSG; for a reduction in systemic arterial and pulse pressures which trigger the reflex is not present during much of the acceleration exposure if the subject is performing an adequate pulmonary effort.

Although never measured after HSG, the catecholamine response associated with acceleration exposure should be sufficient to produce a generalized sympathetic response--note the apparent psychogenic increase in heart rate before HSG exposure (Fig. 6)--which would augment the heretofore considered cardiovascular responses.

M-1/PPB and Intrathoracic Pressure Relationships

The effect of PPB on systemic arterial pressure during HSG exposure was examined in detail, first by Shubrooks (9), and more recently by Leverett et al. (10). Shubrooks (9) compared systolic/diastolic arterial pressures at eye level at the beginning of vision loss (PLL) in the same persons relaxed or performing the M-1 or PPB. As previously reported by Lambert and Wood (20), PLL occurred in relaxed persons with a mean systolic pressure of 50 mm Hg. When the same subjects performed the M-1 or pressure breathed, however, PLL occurred with mean systolic pressures of 28 mm Hg and 36 mm Hg, respectively. Systolic pressures at PLL for the M-1 or PPB were not significantly different. In both instances, systolic pressures where subjects' vision began to dim were statistically significantly less than those found during their relaxed exposure to G. This information suggests that visual criteria at G do not have necessarily the same correlation with eye-level arterial pressure. Apparently PLL in persons doing the M-1 or PPB is a more stressful end-point than in men relaxed at G.

Shubrooks (9) extended his comparative investigation of eye-level arterial responses to PPB and the M-1 into HSG using three subjects. He suggested that, compared with the M-1, less inspiratory fall in eye-level arterial pressure occurred in persons doing PPB. Shubrooks concluded that a significant portion of these arterial pressure responses at HSG, in those persons using PPB, could be attributed to subject training, degree of muscular tensing, and the use of the anti-G suit.

Leverett et al. (10) expanded these HSG/PPB studies using six subjects, and they measured both esophageal (EP) and arterial pressures. During the M-1 maneuver or PPB, fluctuations in EP and AP were observed. They are shown in Figure 13 for three subjects during 8 G exposure. Changes in AP are directly correlated with changes in EP (viz, both in frequency and amplitude). Esophageal and arterial pressures obtained during 1, 6, and 8 G are compared for the M-1 and PPB in Table VI. Fluctuations in EP correlated with inhalation and exhalation, resulting in a cyclic phenomenon which exhibited minimum and maximum values for systolic arterial pressures, pulse pressures, and esophageal pressures--the minimum value occurring during the rapid inspiratory phase of respiration, and the maximum value corresponding with the prolonged period of forced expiration.

Systolic arterial blood pressure and pulse pressure were not affected differently by the M-1 or PPB. Systolic pressures were lower during inspiration than expiration, and each was reduced as the G level increased. Mean systolic pressures at 8 G during inspiration were approximately 20 mm Hg, the arterial level at which central light loss should occur. However, regarding individuals, it was not uncommon during 8 G runs for the systolic arterial pressure to be zero during the inspiratory phase of the M-1, yet the subjects reported no loss of vision. If the subject's rate of inspiration was sufficiently slowed, however, the lower pressures were maintained for longer periods; and then subjects reported visual loss. Of course, PLL occurred when eye-level pressure at all phases of the respiratory cycle dropped to near 30 mm Hg. Usually, the mean systolic blood pressure during expiration at 8 G was approximately 90 mm Hg, which is

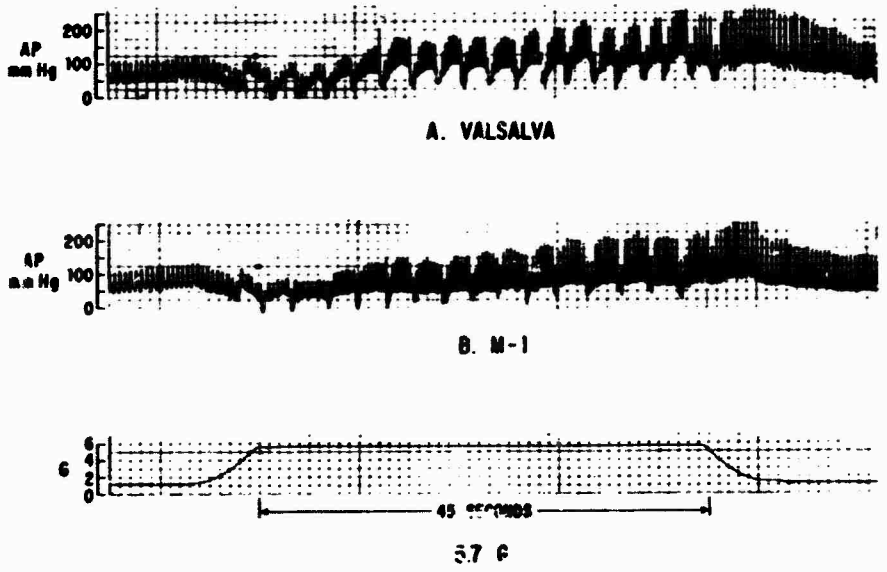


Fig. 12: Arterial pressure (AP) responses at eye-level are compared for the M-1 and L-1 (called a Valsalva in this figure) at 5.7 G with the subject wearing an anti-G suit. (Similar responses are seen at 7 G in Figure 5 of Shubrooks and Leverett (8).)

Table VI: Systolic arterial blood pressure (SAP), pulse pressure (PP), and esophageal pressure (EP), are compared during inspiration (min) and expiration (max) at 1 G, 6 G, and 8 G in persons performing the M-1 or PPB. Shown are means ± standard errors for a group of subjects, the "n" of which is in parentheses (ref 10).

	M-1			PPB		
	1 G	6 G	8 G	1 G	6 G	8 G
SAP:						
Min	101±10.1 (6)	58.3±13.0 (6)	18.4±12.3 (6)	106±7.57 (5)	52.7±14.3 (6)	21.9±19.8 (6)
Max	166±8.21 (6)	135±13.9 (6)	94.8±17.9 (6)	160±11.5 (5)	111±9.01 (6)	90.4±22.1 (6)
PP:						
Min	35.8±5.43 (6)	37.8±7.07 (6)	30.6±2.06 (6)	33.9±5.70 (5)	31.2±9.42 (6)	24.9±10.3 (6)
Max	59.0±9.15 (6)	83.4±7.57 (6)	72.5±10.4 (6)	68.2±3.51 (5)	76.3±3.75 (6)	72.9±13.9 (6)
EP:						
Min	-8.98±5.40 (6)	*** -21.3±1.90 (6)	* -25.6±3.82 (6)	6.17±2.21 (6)	*** 7.03±1.78 (6)	* 4.54±1.86 (6)
Max	** 61.6±3.45 (6)	48.5±6.49 (6)	70.9±5.10 (6)	** 33.2±4.30 (6)	37.8±5.18 (6)	58.4±10.1 (6)
#Differential	* 70.0±8.19 (6)	* 68.6±10.2 (6)	* 96.4±6.04 (6)	* 27.1±5.88 (6)	* 30.8±6.96 (6)	* 59.2±11.3 (6)

#Differential EP = Max EP - Min EP; * = $P < 0.05$; ** = $P < 0.01$; *** = $P < 0.001$

more than sufficient to maintain adequate cerebral and retinal blood circulation. Pulse pressures were lower during inspiration than expiration, but appeared to be unaffected by the G-level.

Esophageal pressure was affected by PPB quite differently than by the M-1. Generally speaking, during inspiration, EP was lower in those persons doing the M-1 than in those doing PPB. Conversely, during expiration, EP was higher in subjects doing the M-1 than in those doing the PPB--resulting in mean differential EP (defined in Table VI) approximately twice as large during the M-1 as during PPB.

As already noted, Shubrooks (9) found less respiratory-induced variations in systemic arterial blood pressure with PPB than with the M-1 at HSG. On the other hand, Leverett et al. (10) were unable to

demonstrate consistently a similar finding (Table VI). According to the Leverett study, three types of arterial pressure responses appeared to be associated with PPB and the M-1, as shown in Figure 13: (a) for subject RZ, less pressure fluctuation with PPB (similar to that reported by Shubrooks); (b) for RS, less pressure variation with M-1; and (c) for HL, similar pressure responses with M-1 or PPB.

Two important differences in these experiments exist which may explain these apparent discrepancies. Shubrooks had fewer yet probably better trained subjects who used 40 mm Hg PPB, whereas the Leverett study used 30 mm Hg PPB. Considering both studies, however, it may be concluded that: (a) PPB can reduce, significantly, the marked variations in arterial pressures at eye-level in specific individuals (viz, RZ in the Leverett study, and subject P in the Shubrooks study); and (b) although this marked reduction in arterial pressure fluctuation is not apparent in other subjects while pressure breathing, PPB is as effective as the M-1 in maintaining eye-level arterial pressure.

Venous Pressure Responses

Leverett et al. (10) attempted to measure venous pressures in the superior vena cava during HSG using three pressure transducers. They felt that their methods eliminated the intrathoracic pressure effect--this effect is substantial at HSG with the subject performing the M-1--which resulted in a "net central venous blood pressure." They were able to identify four specific pressure responses during HSG exposure, relative to cardiac and respiratory cycles qualitatively similar to EP responses. However, interpretation of these data was not possible.

In the same study (10), however, the authors were able to measure lower body venous pressure by using a superficial vein in the region of the ankle. (As noted previously in this text, persons wearing the RAF mini anti-G suit complained of severe calf pain during exposure to HSG--especially at the 6 G level.) It was hypothesized that a direct correlation might exist between venous pressure in the region of the lower leg during G and the occurrence of pain.

Superficial venous pressures at the ankle were measured in five subjects who wore the mini-suit at 1, 3, 6, and 8 G. Continuous pressure recordings obtained before, during, and after acceleration exposures showed the existence of a dynamic pressure rise during the period of increasing G (Fig. 14). It appeared that venous pressure in this region had a two-component basis: (a) M-1 effect--apparent even at 1 G, with the subject performing a maximum M-1; and (b) acceleration-time effect.

Ankle venous pressures (mean ± standard error) were compared for subjects grouped according to their pain responses at 6 and 8 G: no pain, 191 ± 23.2 mm Hg; moderate pain, 384 ± 46.3 mm Hg; and severe pain, 214 ± 19.8 mm Hg. (Persons exhibiting moderate pain had, statistically, significantly higher pressures than those with severe pain; and the ankle venous pressures of persons exhibiting severe pain was not significantly different from that of persons without leg pain at HSG.) Consequently, it appears that a pain:ankle-venous-pressure simple relationship does not exist.

Heart Mass Size Changes

Using radiographic techniques during HSG, Leverett et al. (10) were able to detect a change in heart shadow size relative to the G-level. The relationship between G-level and heart height was reported to be inverse; and, by using each subject's cardiac measurement (n = 31 pairs), a regression analysis was developed showing the following correlation with G:

$$H = 9.68e^{-0.063\ G} \qquad (1)$$

in which:

H = heart height (cm),
G = acceleration level; and,
Correlation coefficient (r) = 0.75; ($P < .01$).

On the other hand, heart width had no statistically significant relationship with G, and on all X-rays appeared to be quite consistent.

Post-acceleration Responses

After HSG exposure, cardiac arrhythmias were found occasionally by both Shubrooks (7) and Leverett et al. (10), as already illustrated in Figure 9, and therefore will not be discussed here.

Two other cardiovascular responses, reported by Leverett et al. (10), occurred during the first minute following HSG: (a) marked sinus arrhythmias; and (b) a gradual increase in systemic arterial pressure, peaking approximately 60 sec post-G exposure (Fig. 15).

The sinus arrhythmia was most apparent after 6 and 8 G, 60 sec exposures. Interestingly, the sinus arrhythmia continued as the post-G arterial pressure overshoot developed, and then subsided as the eye-level arterial pressure returned toward pre-G levels. However, although a "post-G" overshoot occurred in persons performing the M-1 or PPB at 1 G for 60 sec, a marked sinus arrhythmia "post-G" developed only in conjunction with PPB; i.e., sinus arrhythmia was not found in those persons performing the M-1 at 1 G or after 60 sec of 3 G.

Fig. 13: A comparison of arterial (BP) and esophageal (EP) tracings obtained from three subjects who were exposed to 8 G while performing either the M-1 or PPB. RZ, HL, and RS = three subjects for the M-1 and PPB tests (ref 10).

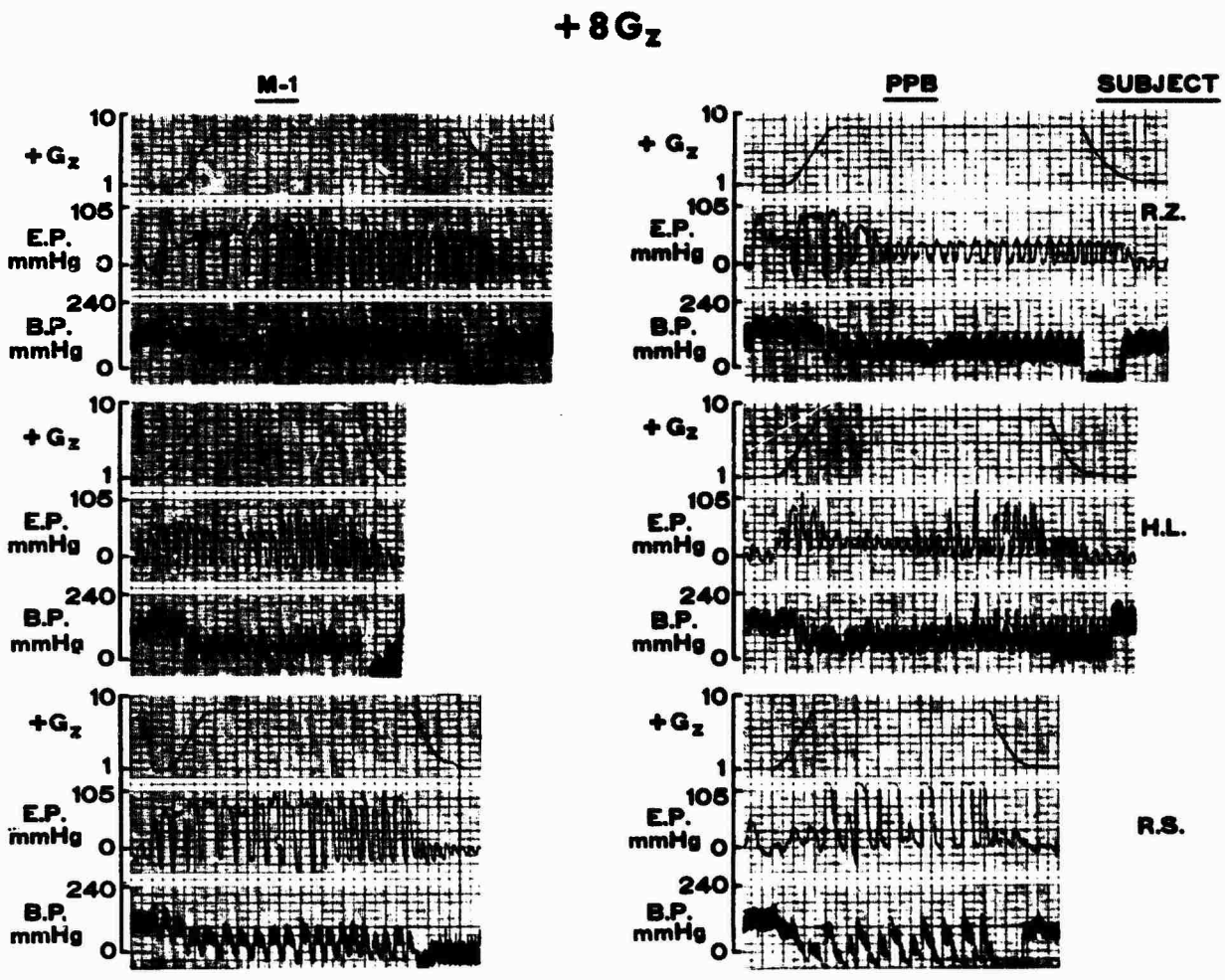
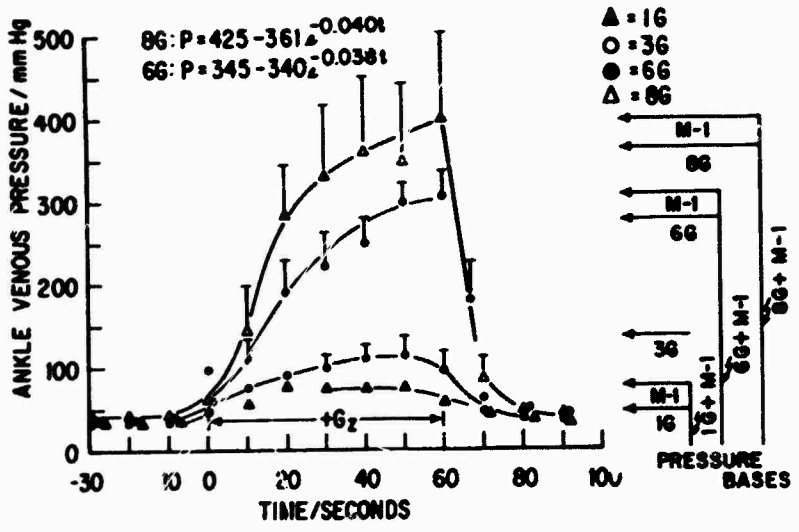


Fig. 14: The effects of time, at various levels of G, on lower body venous pressure (mm Hg) are compared with calculated hydrostatic pressure bases (ref 10).



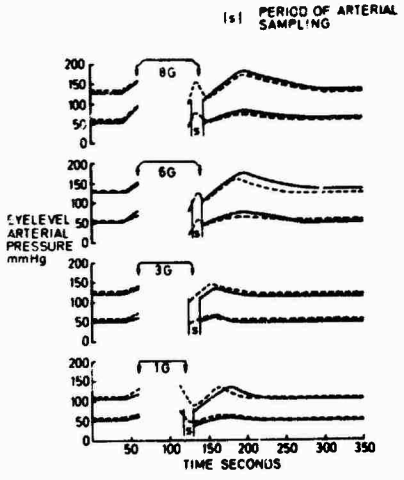


Fig. 15: Systolic and diastolic arterial pressures at eye-level are compared for the various G-levels before and after acceleration exposure. The solid and dashed lines denote 2 separate but similar studies. The time period marked with an "s" immediately post-G indicates arterial blood withdrawal from the subjects (note the section in this text, "Pulmonary and Respiratory Responses of Man to HSG," ref 10).

Because this systemic arterial pressure overshoot was found in persons at 1 G performing the M-1 or PPB and long after cardiovascular induced overshoot had occurred (Fig. 15), Leverett et al. (10) proposed that this post-G rise in arterial pressure may have a metabolic basis--specifically, a function of lactic acid buildup during strenuous physical efforts. Also, the time required to reach a peak was directly correlated to G-level, the longest time being in those situations where the largest buildup of metabolites presumably would occur.

Pulmonary and Respiratory Responses of Man to HSG

The lung is an organ which is particularly susceptible to the effects of HSG. The expected increase in the impairment of gas exchange in the lung of man at HSG can cause severe reduction in oxygen transport to the brain, impairing man's ability to tolerate acceleration. The physiologic effects on the lungs during exposure to lower levels of $+G_z$ acceleration have been examined in detail over the years by many investigators, and these studies have recently been reviewed by Glaister (4). It is generally agreed that the major effects of $+G_z$ on the pulmonary system are airway closure, atelectasis, and altered distribution of ventilation and blood flow resulting in venous admixture and hypoxia. With exposures of man to higher acceleration magnitudes for longer durations, these abnormalities would become more serious; and other pathophysiologic effects (such as altered surface forces, pulmonary edema, and interference with chest wall mechanics) would be expected to assume increasing significance as factors in limiting HSG tolerance. There is also the possibility that disruption of the mechanical integrity of the lung itself might occur.

Pulmonary Function

Until recently (10, 11) little was known of pulmonary gas exchange or blood oxygenation in man exposed to HSG. Michaelson (11) exposed nine male volunteers to levels of +1 G, 3 G, 5 G, and 7 G_z for 45 sec. The subjects wore a standard USAF anti-G suit and breathed air. Continuous measurements were obtained for: end tidal CO_2 tension ($P_{ET}CO_2$); tidal volume (V_T); respiratory rate (f); and heart rate (HR). During the last 20 sec of a 45-sec G exposure, expired gas was collected and a blood sample was withdrawn from the radial artery. The mixed expired oxygen and carbon dioxide gas tensions and the arterial gas tensions (PaO_2, $PaCO_2$) and pH were measured. Furthermore, four of the same subjects were similarly studied, except that 100% O_2 was breathed for 15 min before and during the G exposure.

In Table VII is shown the mean data obtained by Michaelson (11) from air-breathing subjects. Although respiratory rate increased progressively from 1 to 7 G, the increase of V_T at higher levels appears to be limited. This effect could be due, at least in part, to G-induced mechanical limitations on the chest wall; but abdominal compression by the inflating anti-G suit was probably the primary cause.

With the data of Table VII, Michaelson (11) computed the alveolar-arterial O_2 tension gradient $(A - a)O_2$ and the arterial-end tidal CO_2 tension gradient $(a - ET)CO_2$ from which could be derived several indices of pulmonary gas exchange. We emphasize here that the results of these estimates--shown in Table VIII--are only approximations; for it is assumed that the subjects did not attain a true steady-state during G exposure. Although an approximate 3.5X increase occurs in ventilation (11) from 1 to 7 G, very little change in $PaCO_2$ is associated with an increasing G which is consistent with the calculated increase in physiologic dead space (V_D) and the V_D/V_T ratio.

An inverse relationship between the level of G exposure and PaO_2 was apparent in the 9 subjects breathing air (Fig. 16). In a few subjects, the PaO_2 falls to less than 50 mm Hg. The $(A - a)O_2$ gradient (at 7 G) as computed by Michaelson (11) from the mean data of the nine men is 66 mm Hg.

Although neither mixed venous gas tensions nor cardiac output were measured in Michaelson's study (11), he found it possible to estimate the degree of venous admixture ($\dot{Q}s/\dot{Q}t$), which would account for the observed $(A - a)O_2$ gradient when a value for the arterial-mixed venous oxygen difference $(a - \bar{v})O_2$ is assumed. This was accomplished for $(a - \bar{v})O_2$ = 3, 5, and 8 vol% and is shown also in Table VIII.

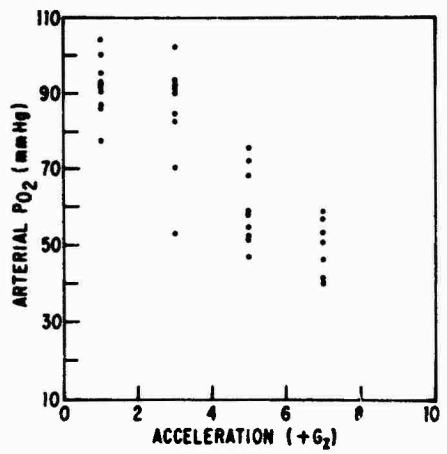


Fig. 16: PaO_2 determined from blood withdrawn over the last 20 sec of 45 sec exposure to G. Each point represents one subject (ref 11).

The effect of breathing 100% oxygen at 1 and 5 G upon several pulmonary indices was determined by Michaelson (11), using 4 subjects (Table IX). In this study, 7 G was not attempted because of subject fatigue. These values are similar to those reported by Barr et al. (41). The difference in calculated $\dot{Q}s/\dot{Q}t$ between the air and oxygen breathing (Tables VIII and IX) suggests that, for a 45-sec 5 G exposure, complete anatomic collapse cannot account for the observed $(A - a)O_2$ gradient in the air breathing subjects. However, the relative contributions of alveoli with low ventilation-perfusion ratios and alveoli that are unventilated but contain trapped gas cannot be distinguished. Since alveoli with trapped air would equilibrate rapidly with the mixed venous blood and behave like anatomic shunts, while blood equilibrating with oxygen trapped in alveoli would remain fully saturated until complete alveolar collapse occurred. Furthermore, since the degree of atelectasis depends on time at G and is highly dependent on alveolar gas composition, the physiology of the air-filled lung is not directly comparable to that of the oxygen-filled lung. This observation could also partially explain the apparent conflict among various reports, as reviewed by Glaister (4), some of which have suggested that no difference occurred in $\dot{Q}s/\dot{Q}t$ during air as compared with oxygen breathing, while others have shown large values for $\dot{Q}s/\dot{Q}t$ during both air and oxygen breathing. As time at G increases while O_2 breathing, O_2 will continue to be absorbed from units distal to occluded airways, and $\dot{Q}s/\dot{Q}t$ would increase as alveolar collapse progressed. This increased shunt may account more fully for the $(A - a)O_2$ observed during air breathing.

M-1/PPB Effects on Arterial Blood Gas and pH

Leverett et al. (10) examined the effects of HSG and the M-1 and PPB upon arterial blood gas and pH by subjecting six volunteers to 60 sec of 1, 3, 6, and 8 G (Tables X and XI). Each man maintained vision at HSG, using the M-1 or PPB. The 1 G exposure consisted of 60 sec of maximum M-1 or PPB effort by the subject--an effort similar in degree to that performed at 8 G in order to maintain vision. The subjects wore

Table VII: Mean values ± standard deviation (in parentheses) of 9 men breathing air and exposed to 45 sec of $+G_z$ (ref 11).

	1 G	3 G	5 G	7 G
PaO_2	91.6 (7.9)	84.7 (14.8)	60.2 (9 9)	50.1 (7.3)
$PaCO_2$	35.0 (4.1)	32.0 (4.0)	32.1 (3.0)	33.2 (2.0)
pH	7.422 (0.022)	7.422 (0.036)	7.444 (0.025)	7.418 (0.034)
$P_{ET}CO_2$	33.6 (3.5)	27.3 (4.6)	20.2 (3.6)	15.8 (3.3)
P_EO_2	119.2 (2.8)	127.2 (3.8)	132.2 (4.5)	136.0 (2.3)
P_ECO_2	22.7 (1.9)	20.0 (1.9)	15.5 (3.5)	12.8 (2.8)
f	18.6 (3.0)	23.4 (4.8)	32.6 (7.5)	38.9 (13.2)
V_T	0.68 (0.11)	0.93 (0.21)	1.20 (0.31)	1.13 (0.44)
HR	75 (7)	109 (18)	147 (22)	164 (19)

Table VIII: Calculated values from the data of Table VII.

	1 G	3 G	5 G	7 G
P_AO_2*	101.3	112.8	114.8	116.3
$(A - a)O_2$	9.7	28.1	54.6	66.2
$(a - ET)CO_2$	1.4	4.7	11.9	17.4
V_D	.200	.307	.579	.551
V_D/V_T	.35	.38	.52	.52
$\dot{Q}s/\dot{Q}t$ (3)†	---	11.2	33.8	49.2
$\dot{Q}s/\dot{Q}t$ (5)†	2.9	7.1	23.4	36.8
$\dot{Q}s/\dot{Q}t$ (8)†	---	4.5	16.1	26.7

* Alveolar oxygen tensions were computed by Michaelson (ref 11) from values of Table VII using the alveolar gas equation.

† Assumed vol % $(a - \bar{v})O_2$.

a standard USAF anti-G suit. A blood sample was obtained from a radial arterial cannula immediately after termination of a 60-sec exposure--during the period of deceleration (note Fig. 15). Immediately after blood collection, PaO_2 and $PaCO_2$ and pH were measured.

Not all of the subjects in the 8 G group, however, tolerated this level of acceleration for the entire 60 sec--the tolerance range being 8 sec to 1 min (Tables II and III). Blood was withdrawn during the deceleration period at the termination of the 8 G exposure period, regardless of the duration of exposure. Since a wide range of tolerances existed, an estimate could be made of the change in arterial oxygen tension as a function of exposure duration at 8 G (Fig. 17). If the mean arterial PaO_2 of the 1 G group is considered as approximating 0 time at 8 G (this estimate ignores the acceleration onset time since approximately 7 sec of acceleration increase is necessary for the centrifuge to reach 8 G), a rapid reduction in oxygen tension occurs during the first 20 sec exposure to 8 G. However, continued acceleration exposure for an additional 40 sec causes little additional reduction in PaO_2. The M-1 or PPB did not affect PaO_2 differently at 8 G. The effect of time at 8 G upon PaO_2, using both PPB and M-1 data, is best described mathematically by:

$$PaO_2 = 104T^{-0.196} \quad (2)$$

in which:

PaO_2 = arterial oxygen tension (mm Hg);
T = time at 8 G (sec); and
$r = 0.90$; $P < 0.01$

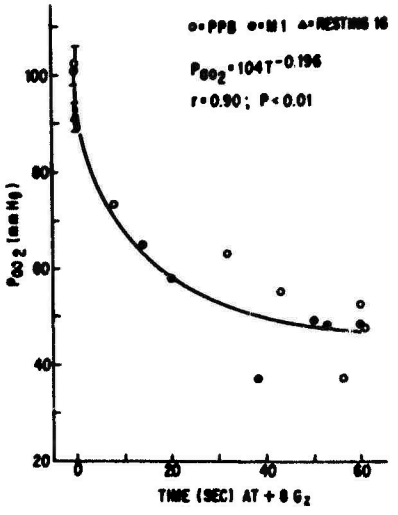


Fig. 17: Effect of exposure time at peak 8 G on PaO_2 for both M-1 and PPB subjects (ref 10). (The resting 1 G value is from Michaelson, ref 11.)

Exposure time during sustained high G is obviously important in determining arterial oxygen tensions for a given G-level. Consequently, a mean value of PaO_2, representative of 8 G in a sustained acceleration study, should include only those values obtained after the subject had been at peak G for some period of time. A minimum of 50 sec exposure to 8 G was considered by Leverett et al. (10) as representative of a HSG exposure, because PaO_2 values appeared to have reached a "near-steady state" after 50 sec of 8 G.

Arterial blood gas-pH parameters (group means ± standard errors) obtained from persons at various G levels (performing the M-1 as necessary) are listed in Table X. The PaO_2 values are compared in Figure 18 with arterial oxygen tensions of Michaelson (11) shown in Figure 16. The PaO_2 values from both studies are quite similar in regard to G effect (except at 3 G), consequently all PaO_2 data were combined (except Leverett "M-1" and Michaelson "3 G") and the following PaO_2 relationship to G was mathematically determined:

$$PaO_2 = 97.5\, e^{-0.093\, G} \quad \ldots\ldots\ldots \quad (3)$$

in which:

PaO_2 = arterial oxygen tension (mm Hg);
G = level of HSG; and,
r = 0.996; P < .01.

The higher PaO_2 at 3 G (obtained by Michaelson) suggests that the rapid reduction in PaO_2 found at HSG (Fig. 17) apparently does not occur at the lower 3 G level. The higher value of PaO_2 at 1 G (M-1 in Fig. 18), in the Leverett study, can be attributed to hyperventilation during the M-1 maneuver. The M-1 was <u>not</u> performed during collection of the 1 G data of Michaelson. This assumption is consistent with the different $PaCO_2$ values obtained at 1 G in the two studies (Tables VII and X).

A comparison of the effects of HSG on PaO_2 during the M-1 maneuver with the PaO_2 during PPB is of practical interest (Fig. 19); for PPB, from a cardiovascular standpoint, is an attractive alternative to the M-1 maneuver for enhancing HSG tolerance (9, 10).

The PaO_2 of persons during PPB is significantly greater than that obtained from those who did not use PPB at 3 G and used the M-1 at 6 G; but no differences in PaO2 were noted at 8 G. These observations could possibly be attributed to an increased oxygen requirement during the M-1 maneuver, as compared with PPB at these levels, and/or a modification by PPB of the G-induced changes in the lung. Because, according to Glaister (4) the airways in the dependent portions of the lung have been shown to collapse during $+G_z$, the ventilation-perfusion inequality is exaggerated. The result is a greater degree of venous admixture than might be explained by the G-induced redistribution of blood alone. An increase in the functional residual capacity (FRC) during PPB would tend to minimize this dependent airway closure. A higher FRC would also reduce the effect of alveolar surface forces and thus limit closure of terminal lung units. The effect of PPB on the FRC during $+G_z$ could also account, at least in part, for the similar values of PaO_2 for the M-1 maneuver and PPB observed at 8 G; for, at 7 G, inflation of the anti-G suit appears to limit lung expansion (11). FRC was not measured in the study by Leverett et al. (10); and the possible effects of PPB on FRC, as it affects PaO_2 remain speculative. Furthermore, at 8 G the increase in ventilation-perfusion inequality may assume greater significance as compared with the beneficial effects of PPB, although this possibly seems unlikely because of the apparent exponential relationship between PaO_2 and G (Eq. 3). The beneficial effects of PPB at HSG also may be limited by a reduction in cardiac output, an overexpansion of normal areas of lung, and an increase in the vertical non-uniformity of blood flow in the lung. Nevertheless, the higher values of PaO_2 observed at 3 G and 6 G during PPB should give a physiological advantage in terms of oxygen transport where transient levels of 7 G to 10 G may be reached from a 3 G to 6 G plateau.

According to Michaelson (ref 11, Table VII), Leverett et al. (ref 10, Tables X and XI), HSG had little effect on arterial CO_2 tension. (The reasons have already been discussed.) The $PaCO_2$ at +1 G_z during the M-1 maneuver is slightly but significantly ($P < 0.05$) lower than the $PaCO_2$ at 3 G and 6 G, probably the result of hyperventilation with a relatively normal physiologic dead space. Unfortunately, interpretation of the $PaCO_2$ data from the Leverett study is difficult, because measurements of ventilation were not made.

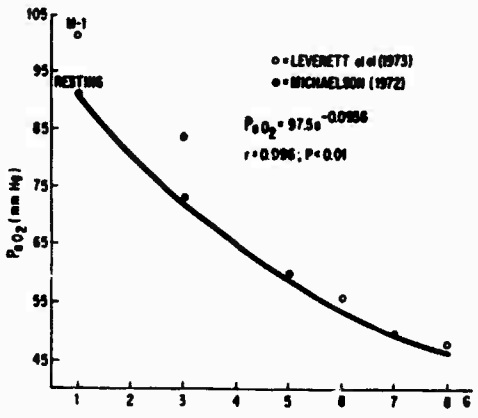


Fig. 18: The PaO_2 data of Michaelson (ref 11) are compared with PaO_2 values of Leverett et al. (ref 10). The equation considers all points except Michaelson at 3 G and 1 G (M-1) as explained in text.

Table IX: Mean values ± standard deviation (in parentheses) of 4 men breathing 100% oxygen and exposed to 45 sec of 1 or 5 G (ref 11).

	1 G	5 G
PaO_2	616 (21)	570 (48)
$PaCO_2$	35.8 (2.6)	39.2 (2.0)
pH	7.401 (.028)	7.418 (.032)
$P_{ET}CO_2$	31.0 (2.4)	21.6 (4.3)
f	17.7 (2.2)	24.1 (13.2)
V_T	.60 (.15)	.87 (.24)
HR	85 (7)	158 (25)
P_AO_2	663	660
$(A-a)O_2$	47	90
$(a-ET)CO_2$	4.8	17.6
$\dot{Q}s/\dot{Q}t$ (3)*	---	7.1
$\dot{Q}s/\dot{Q}t$ (5)*	3.1	4.4
$\dot{Q}s/\dot{Q}t$ (8)*	---	2.8

* Assumed vol % $(a-\bar{v})O_2$

Table X: Arterial O_2 and CO_2 tension and pH (group mean ± standard errors in parentheses) are shown for 6 men performing the M-1* and exposed to various levels of G for 60 sec** (ref 10).

	1 G	3 G	6 G	8 G
PaO_2	101.3 (3.7)	73.1 (5.5)	56.3 (6.0)	48.3 (0.33)
$PaCO_2$	27.4 (2.1)	31.1 (1.1)	32.0 (1.1)	29.7 (2.0)
pH	7.43 (0.02)	7.44 (0.01)	7.44 (0.01)	7.44 (0.02)

* Subjects were required to perform a maximum M-1 during 1 G exposure. The 3 G exposures did not require the M-1.

** 8 G means consist of only those subjects (n = 3) who were capable of tolerating at least 50 sec of 8 G.

Although no direct measurements of SaO_2 were made in the above described studies, reasonable estimates of the effect of HSG on SaO_2 can be made from PaO_2; for both studies showed very minimal change in $PaCO_2$ and pH with increasing G. Leverett et al. (10), using PaO_2 data, calculated the SaO_2 and were able to relate the saturation changes; with time at 8 G (Eq. 4) and with levels of G (Eq. 5): i.e.,

$$SaO_2 = 96.4 - 0.197T \quad \ldots\ldots\ldots \quad (4)$$

in which:

SaO_2 = arterial saturation (%);
T = same as Eq. 2; and,
r = 0.73; P < 0.01

and:

$$SaO_2 = 99 - 1.59G \quad \ldots\ldots\ldots\ldots \quad (5)$$

in which:

SaO_2 = same as Eq. 4;
G = same as Eq. 3; and,
r = 0.70; P < 0.01.

A linear relationship between SaO_2 and G would be expected for the exponential fall in PaO_2 with increasing G or time at 8 G--refer to equations (2) and (3)--would be significantly altered by the sigmoidal shape of the oxyhemoglobin saturation curve. Accordingly, arterial saturation at HSG may continue to decrease at a constant rate relative to G-level or time at HSG.

Glaister (4) also found a rectilinear relationship between SaO_2 and levels of sustained $+G_x$. As in the Leverett study (10), a reduction in saturation began at about 3 G. The rate of fall of saturation per $+G_x$ appeared to be approximately 3%, however, whereas 1.59% (slope of Eq. 5) fall in saturation per G accompanies increasing levels of $+G_z$ (i.e., approximately 50% less than at $+G_x$).

Electroencephalograms (EEG) of Man at HSG

The electroencephalogram (EEG) is especially appropriate for use on the centrifuge. This natural electro-response, much like the ECG, only has to be amplified (not changed) and may be easily monitored at some distance from the subject. The EEG profile, a qualitative measurement of brain activity, appears to be directly correlated with several cardiovascular, respiratory, and performance parameters obtained during HSG.

Berkhout et al. (12) exposed eight men to 45 sec of +4.5 G, 6 G, and 7 G_z. The 4.5 and 6 G exposures were repeated six times over a 15 min period. They found the EEG to be altered during G exposure and for 30 sec post-G, even in the absence of visual blackout, and attributed this fact to compensatory cardiovascular changes associated with the accommodative physiology of G. These EEG changes were not pathologic in nature and were noncumulative with repeated G exposures; however, by using the EEG it was possible to identify the higher G levels as more "stressful." Berkhout et al. (12) concluded that human performance levels at HSG are not compromised by the physiology associated with CNS changes which occur at G.

HSG in Combination with Other Stressors

Cigarettes and ethanol are frequently used during off-duty hours by pilots of high performance aircraft. Smoking would probably have its greatest effects on the lung, and alcohol consumption is commonly known to reduce performance. These specific effects, in combination with HSG have been examined recently by Michaelson (14) and Burton and Jaggars (13).

Effect of Cigarette Smoking on the Mechanical Properties of the Lung After Exposure to HSG

Michaelson (14) exposed 11 volunteers using standard USAF anti-G suits to HSG (7 to 9 G for 45 sec). Immediately afterwards--60 and 90 sec post-G--he measured their vital capacity (VC) using a dry seal spirometer mounted in the gondola of the SAM human centrifuge. Of the subjects, 4 were cigarette smokers who used at least one pack per day, and 7 did not use any form of tobacco.

Post-HSG measurements, examined as percent change from pre-G data, are compared in Table XII. The seven nonsmokers had a small -3.1% reduction in VC at 60 sec, and a somewhat greater fall of -5.4% at 90 sec post-G exposure--not significantly different; $P > 0.05$. On the other hand, the four smokers had a group mean decrease of VC of 7.9% at 60 sec, and 12.3% at 90 sec ($P < 0.02$) compared to pre-G values.

The observed changes in VC, although small, are remarkable; for previous studies, reviewed by Glaister (4), in subjects breathing air did not show a significant fall in VC up to levels of +6 G_z. These findings suggest that exposure to levels of 7 G to 9 G for 45 sec combined with anti-G suit inflation (and especially in the smokers) is sufficient to produce regional compression atelectasis and/or airway closure without having to invoke rapid absorption of oxygen distal to occluded airways. Furthermore, in both the nonsmokers and the smokers ($P < .02$), the reduction in VC was greater at 90 sec than at 60 sec. This implies that progressive collapse or reduction in compliance may be occurring at 1 G after HSG exposure--possibly due to altered surface forces in alveoli or very small terminal airways.

Because of these findings, it might be expected that acceleration atelectasis, which has been observed at lower +G_z levels during oxygen breathing, would be grossly exaggerated during exposure to HSG--especially in those persons who smoke cigarettes. Interestingly, U.S. Navy pilots of high performance aircraft (breathing 100% oxygen) who smoke cigarettes are more prone to atelectasis than their nonsmoking counterparts (42).

It was necessary to determine if muscular fatigue, induced by straining during the M-I maneuver, contributed to the observed changes in VC. Accordingly, Michaelson subjected each man, on a separate day, to a series of maximal effort M-I maneuvers at +I G_z for 45 sec at a frequency similar to that of their M-I during the HSG exposures. VC was measured before, and at, 60 sec and 90 sec after the series of M-I maneuvers. No significant difference existed between the pre-M-I and post-M-I measurements (i.e., the mean 60 sec and 90 sec VC were slightly greater than the pre-M-I).

Task Performance at HSG After Alcohol Ingestion

The effects of the combination of various oral doses of ethyl alcohol and various levels of sustained G upon performance has been determined by Burton and Jaggars using a "target-tracking task." Eight adults (7 males and 1 female) drank orange juice mixed with various doses (0, 0.5, 1, 2, or 3 oz) of 95% ethyl alcohol. After a wait of 1 hour, gms percent of blood alcohol (B/A) was determined by breath analysis and the subject was exposed to a series of 7 sec tasks during 45 sec accelerations of 1, 3, 4, 5, and 6 G.

Performance was quantified as the time required to "hit" an electronic target with electronic "bullets." The effects of G and/or alcohol consumption and B/A upon performance were measured as the percent reduction from 1 G without alcohol. The combination of the higher alcohol and acceleration levels resulted in a potentiated reduction in performance. A 9.8% reduction per G in performance was noted per 0.10 B/A in the

range of 1 G through 6 G. Billings et al. (43) reported the occurrence of "major" and "catastrophic" pilot errors using subjects flying aircraft and with an overall reduction in task performance at 1 G of only 2%.

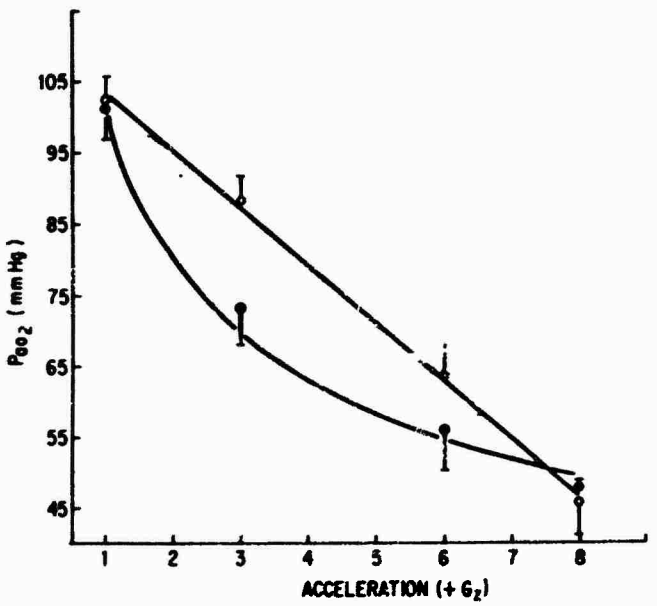


Fig. 19: PaO_2 as a function of G, comparing the M-1 (solid circles) with PPB (open circles). (Mean values at 3 and 6 G are significantly ($P < .05$) different for M-1 and PPB (ref 10).)

Table XI: Arterial O_2 and CO_2 tensions and pH (group means ± standard errors, in parentheses) are shown for 6 men using PPB and exposed to various levels of G for 60 sec* (ref 10).

	1 G	3 G	6 G	8 G*
PaO_2	102.5 (3.4)	88.0 (3.6)	64.0 (5.4)	46.0 (4.7)
$PaCO_2$	27.7 (1.4)	29.3 (1.4)	29.9 (0.9)	29.0 (2.0)
pH	7.45 (0.02)	7.45 (0.02)	7.46 (0.01)	7.46 (0.01)

* 8 G means consist of only those subjects (n = 3) who were capable of tolerating at least 50 sec of 8 G.

Table XII: The mean percent change in measured vital capacity (VC)* at 1 G after exposures to HSG levels of 7 to 9 G for 45 sec is shown for each subject. The subjects are grouped according to cigarette smoking habits (ref 14).

Nonsmokers	60 sec†	90 sec†	No. of exposures to HSG	Avg No. of M-1 per exposure
RS	- 0.4	- 0.8	4	19
HP	+ 3.3	+ 2.0	5	16
SP	+ 3.6	+ 1.4	3	23
RB	-11.1	-13.2	3	18
RG	+ 2.8	+ 0.8	3	24
BS	-12.3	-11.2	3	21
WC	-10.9	-18.3	4	19
Mean	- 3.6	- 5.4		
SEM	2.8	3.2		

Smokers	60 sec†	90 sec†	No. of exposures to HSG	Avg No. of M-1 per exposure
EB	- 4.2	- 5.8	4	15
VL	- 5.2	-16.9	4	14
WW	-15.4	-15.6	4	20
RK	- 6.6	-10.9	4	14
Mean	- 7.9	-12.3		
SEM	2.6	2.1		

* % change in VC = $\frac{VC\ (pre\text{-}G) - VC\ (post\text{-}G)}{VC\ (pre\text{-}G)} \times 100$

† Time after HSG exposure that VC was measured.

SUMMARY

In studies at USAFSAM, man has tolerated +9 G_z for 45 sec and +8 G_z for 60 sec. Physiologic changes and tolerance limits were the principal purposes of these studies. Man appears to be able, temporarily, to accommodate physiologically to an HSG environment without the development of apparent pathologic sequelae; although, as evidenced by the results of the pathology experiments using the miniature swine, HSG is capable of inflicting pathologic changes without gross functional compromise (viz, pathologic occurrence is not obvious).

The HSG environment to which man may now be subjected is quite unique compared to other types of severely altered environments. That is to say, a person cannot truly protect himself against HSG. Protection against most adverse environmental alterations usually involves the establishment of a "mini-ambient" environment about man, physically preventing the occurrence of significant changes; e.g., hypobaric environment (increase pressures or relative concentrations of specific gasses); high or low temperature (normalize temperature); or radiation (shield against the rays), etc. Changes in acceleration, however, cannot be so physically ameliorated (i.e., G-shields do not exist, nor may we alter the "concentration" of Gs present).

Consequently, man puts on an anti-G suit and by sheer physical effort, by "pulling himself up by his bootstraps," elevates his eye-level arterial pressure so that he can maintain vision. However, as evidenced by eye-level arterial pressure measurements during HSG, brief periods of time exist--during the inspiratory phase of the M-1 or L-1--when the arterial pressures are extremely low and pulse pressures are reduced. Little is known regarding such cardiac physiology at this time.

Eye-level arterial pressure tracings comparing man and the miniature swine (both wearing anti-G suits) at 7 G are remarkably similar (Fig. 20); and at 9 G the pig suffers from subendocardial hemorrhage. The patho-physiology of subendocardial hemorrhage is not well known, nor is it possible for pathologists to predict the cardiac tissue response resulting from repeated occurrences of subendocardial hemorrhage.

It is important, therefore, at this time in the development of HSG research, that "acceleration tolerance" be clearly defined as different from "acceleration protection" regarding human exposure to HSG. Acceleration tolerance versus acceleration protection at HSG was recently reviewed by Burton and Krutz (44). They examined heart rate, esophageal pressure, and arterial oxygen tension as criteria of physiologic protection; i.e., the less these parameters were changed at HSG from established pre-G values--comparing various methods of increasing G tolerance--the greater was the protection offered by the respective anti-G method. They concluded that the reclined seat (65°) and PPB were the only methods presently known to significantly protect man at HSG. The standard USAF anti-G suit, although helping man attain HSG (increased acceleration tolerance), offered no significant protection during exposure to HSG. The protection offered with the reclining seat has both cardiovascular and energetic bases; and PPB appears to offer G protection via the pulmonary (gas exchange) system, coupled with less energy expenditure.

Also, at HSG (+G_z) man becomes extremely fatigued and his performance deteriorates. The reduction in performance appears to be exponentially related to G intensity; and above 5 G, where man must begin to L-1 or M-1, performance rapidly declines (Fig. 21). This deterioration in performance, however, is not related to impaired brain function according to the study of Berkhout et al. (12).

HSG is clearly an environment which is quite restrictive regarding human limitations, i.e., a physiologic steady-state is not possible. Although man's tolerance to +G_z was not exactly determined, several areas regarding body functions were identified which suggest rapidly approaching physiologic limitations: (a) high heart rate, (b) reduction in SaO_2, (c) cardiac arrhythmia, and (d) subject fatigue.

Heart rates of 160 to 200 as found in HSG studies can continue for several minutes without severe cardiovascular compromise. The occurrence of cardiac arrhythmias is common at HSG. They usually are not of serious types; but with a continued high heart rate, rapidly declining SaO_2, and the possibility of endocardial damage, serious heart rhythm disturbances could begin to occur. Considering Eq. 4, SaO_2 appears to be approaching a limit when consciousness cannot continue; viz, if a saturation of 60% is used as a conscious limit, then a person will reach this point (values extrapolated using Eq. 4) after approximately 3 min at 8 G.

It is doubtful, however, that subjects could manage 3 min of 8 G without first becoming fatigued beyond the point of continuing the conscious physical efforts (M-1, L-1, or PPB) necessary to maintain vision. Therefore, fatigue appears to be the critical factor regarding human limitations to HSG. This suggests therefore that the physiology of energetics and physical training (as a method to counteract fatigue) are to become increasingly important in HSG tolerance studies.

Fatigue as a determinant of human tolerance to HSG may be eliminated through the use of the 65° reclining seat, however according to Glaister (4), desaturation occurs during high sustained +G_x (HSG_x) exposure. Consequently, arterial desaturation (hypoxemia) appears to be the limiting factor in persons using the reclining seat and exposed to HSG.

The effects of HSG are marked in terms of gas exchange and arterial hypoxia, and could be a serious limiting factor in performance. Still, many questions remain. The time course of hypoxia at HSG must be

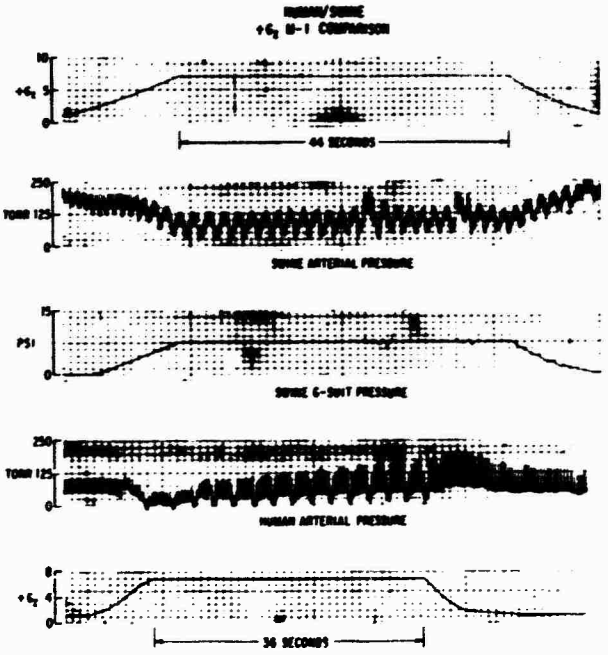


Fig. 20: Eye-level arterial pressures at +7 G_z are compared for man and the miniature swine (ref 17).

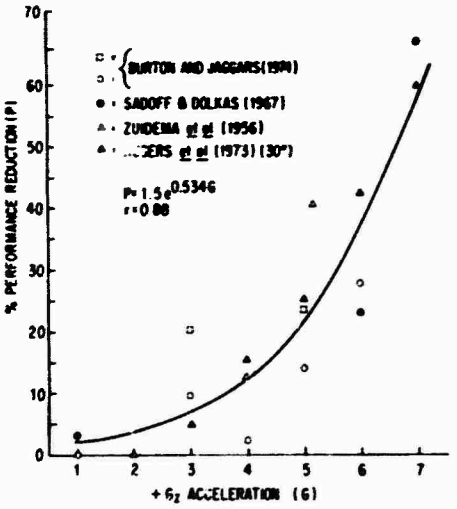


Fig. 21: The effect of sustained $+G_z$ (G) on percent reduction in performance (P) comparing several acceleration performance studies (ref 13, 16, 47, 48).

further explored. The relative contributions of atelectasis, trapped gas and ventilation-perfusion inequality to G-induced hypoxia will be difficult to evaluate until measurements of cardiac output and mixed venous gas tensions are available. Future studies will include both pulmonary gas exchange and mechanics during HSG while the subjects are breathing various combinations of oxygen and inert gas in order to determine an optimum gas mixture, which would both provide for the increased oxygen during HSG and minimize the development of atelectasis.

Throughout this review, the fact becomes apparent that the human pathophysiology of HSG is largely unknown. Unfortunately, scientific progress will be slow for three reasons: (a) HSG is potentially harmful to man, therefore, the scientific approach must be cautious. (b) The physiology of G is dynamic, frequently requiring continuous collection of data (if the values are to be meaningful) by rapidly responsive instruments; and much of this equipment presently is not available to the scientific community. (c) The investigator cannot share the environment with the experimental subject. Consequently, the collection of data must be accomplished at a distance from the subject, thus frequently complicating physiologic studies.

The voluntary informed consent of the subjects used in this research was obtained in accordance with AFR 80-33.

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